Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.2749-19
Intern Med 58: 2887-2890, 2019
http://internmed.jp

【 CASE REPORT 】

Rhabdomyolysis with Acute Kidney Injury Caused by Bilateral Iliopsoas Hematoma in a Patient with Atrial Fibrillation

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Abstract:
Rhabdomyolysis is a relatively common and life-threatening disease that is sometimes complicated by acute kidney injury (AKI). Several causes have been reported, divided into traumatic and non-traumatic causes. We herein report a patient with rhabdomyolysis with AKI caused by bilateral iliopsoas hematoma. This patient had atrial fibrillation that was poorly controlled with warfarin, and bilateral iliopsoas hematoma was caused by turnover without a history of high-energy injury. Treatment with the rapid neutralization of warfarin improved his clinical condition without complications. We should pay close attention to episodes of turnover among elderly patients receiving anticoagulant therapy.

Key words: rhabdomyolysis, acute kidney injury, bilateral iliopsoas hematoma, turnover

(Intern Med 58: 2887-2890, 2019)
(DOI: 10.2169/internalmedicine.2749-19)

Introduction

Rhabdomyolysis is a potentially life-threatening disease that can lead to acute kidney injury (AKI). It is defined as the dissolution of skeletal muscle characterized by the leakage of muscle-cell contents, including electrolyte, myoglobin, and other sarcoplasmic proteins (1). It is mainly caused by trauma or non-traumatic factors, such as side effects of statins and infection. Patients with rhabdomyolysis who are complicated by AKI may require renal replacement therapy for renal indications, such as hyperkalemia, acidosis, or fluid overload not responding to medical therapy (2, 3), and the prognosis of these patients is considered worse than that for patients without AKI (4).

Iliopsoas hematoma is a rare cause of rhabdomyolysis. Its etiology is mainly associated with anticoagulant therapy usage, hematologic diseases like hemophilia, and complications after orthopedic surgery, including total hip arthroplasty (5). In the present case, a patient with poorly controlled warfarin fell onto his back and became complicated with bilateral iliopsoas hematoma; unfortunately, the bilateral iliopsoas hematoma caused rhabdomyolysis with AKI.

This case highlights the need to recognize unusual causes of rhabdomyolysis in patients receiving warfarin therapy.

Case Report

A 70-year-old man with a medical history of atrial fibrillation (Af) on warfarin was emergently brought to the hospital due to dysstasia with worsening bilateral thigh pain from falling onto his back a week earlier. He denied a history of a high-energy accident, like a high-altitude fall.

Over his clinical course as an outpatient, no fever up was seen. His blood pressure, heart rate, and body temperature were 99/84 mmHg, 75/min, and 36.6℃, respectively. A blood analysis showed elevation of creatine kinase (17,504 U/L) and serum creatinine (1.71 mg/dL) levels and an increase in the international normalized ratio of prothrombin time (PT-INR) (6.42). The level of serum hemoglobin (Hb)

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Received: January 26, 2019; Accepted: April 1, 2019; Advance Publication by J-STAGE: June 27, 2019
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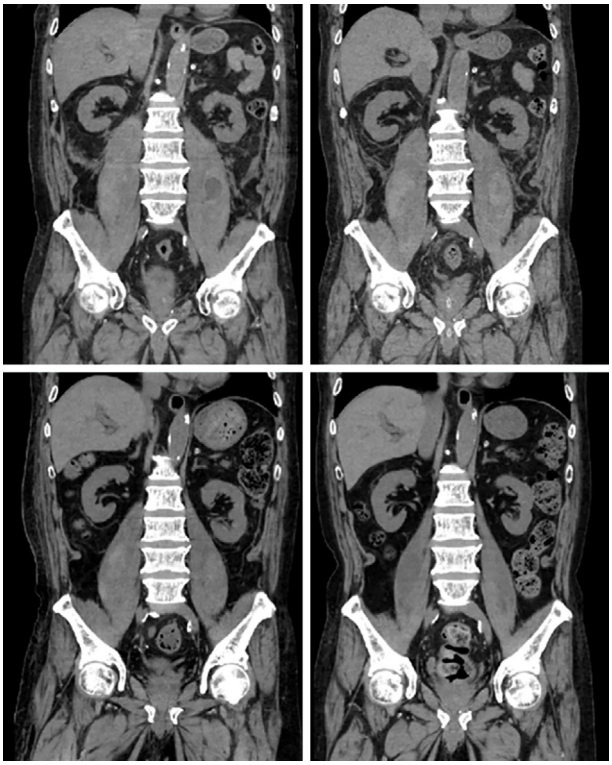

Figure 1. Computed tomography showed a large hematoma in the bilateral iliopsoas. The volume of the hematoma decreased gradually after the administration of four-factor prothrombin complex concentrates. The left upper (Day 1); right upper (Day 3); left lower (Day 11); right lower (Day 24).

(10.5 g/dL) was decreased compared with his usual level as an outpatient (approximately 12.0 g/dL). A urinalysis showed a positive result of occult blood (3+) and some red blood cell urinary sediment (5-9/H). These results suggested that he was suffering from rhabdomyolysis with AKI.

Computed tomography (CT) showed a massive space-occupying lesion (SOL) in the bilateral iliopsoas, suggesting the possibility of iliopsoas hematoma or abscess (Fig. 1). We therefore suspected that he was suffering from rhabdomyolysis with AKI caused by bilateral iliopsoas hematoma or bilateral iliopsoas abscess.

We promptly administered meropenem hydrate (1.0 g), tranexamic acid (1.0 g), and menatetrenone (10 mg). His condition additionally suggested that he might require hemodialysis in the clinical course. Therefore, he was transferred to Mito Saiseikai General Hospital (MSGH), a tertiary emergency medical facility. He was admitted to the critical care unit of MSGH to receive intensive treatment and was administered four-factor prothrombin complex concentrates (4FPCC) (2,000 IU) and fluid infusion without antibiotics or blood transfusion. Massive fluid infusion was administered in order to maintain his urine volume at ≥0.5 mL/kg/h. An arterial blood gas analysis was performed every two hours to monitor changes in his serum potassium levels and the presence of acidosis. Fortunately, his results showed no hyperkalemia or metabolic acidosis.

Follow-up CT showed no increase in the volume of SOL in the bilateral iliopsoas, and the PT-INR recovered to 1.2 after the administration of 4FPCC (Day 2). His serum creatine level also improved almost to the normal level (1.07 mg/dL). He was therefore transferred back to our hospital for further treatment (Day 3) (Fig. 1).

Magnetic resonance imaging (MRI) showed no evidence of iliopsoas abscess but did show findings consistent with iliopsoas hematoma (Day 6) (Fig. 2). Therefore, we ultimately diagnosed him with bilateral iliopsoas hematoma. He started rehabilitation after follow-up CT showed no evidence of an increase in the volume of the bilateral iliopsoas hematoma (Day 11) (Fig. 1). Subsequently we prescribed edoxaban tosilate hydrate (60 mg) for Af. The patient's incorrect dosage of the prescribed medicine was considered to have caused the problematic effect of warfarin. As a result, we decided to prescribe edoxaban tosilate hydrate, which has a once-daily administration and is associated with fewer hemorrhagic complications.

After undergoing therapy, he began to ambulate and recover slowly. He was able to be discharged without recurrence of iliopsoas hematoma or any complications (Day 37) (Fig. 3). Given that his medical history and family history did not indicate any risk of hemophilia or other hematologic diseases, his incorrectly taking warfarin over the prescribed dose and fall were deemed the main causes of his bilateral iliopsoas hematoma. Therefore, we discussed his post-discharge lifestyle with caregivers and his family in order to ensure he would take his medicine regularly.

Discussion

Rhabdomyolysis is a relatively common disease encountered in the emergency department setting. Several causes of rhabdomyolysis are reported, being mainly divided into traumatic and non-traumatic causes (6). Crush syndrome is a well-known traumatic cause, but there are a variety of non-traumatic causes as well, such as a hereditary predisposition, infection, electrolyte imbalance, and side effects of medication (6, 7). Trauma with muscle damage is often reported as a cause of rhabdomyolysis, and iliopsoas hematoma is a relatively rarely seen cause.

Rhabdomyolysis complicated by AKI requires careful treatment, including intensive care, due to its severity. Iliopsoas hematoma is closely associated with anticoagulant therapy usage and hematologic diseases, like hemophilia (8, 9), and in patients taking warfarin therapy incorrectly, iliopsoas hematoma can increase the volume and spread widely, sometimes bilaterally (10). In the worst cases, patients with iliopsoas hematoma may require blood transfusion which can sometimes lead to mortality (10, 11). Therefore, rapidly neutralizing warfarin is necessary to prevent widespread bleeding as soon as possible. If there is a way to neutralize an anticoagulant exacerbating iliopsoas hematoma, then appropriate measures should be taken as quickly as possible.

Rhabdomyolysis is a relatively common disease but can lead to a fatal outcome (4). Therefore, it was very important

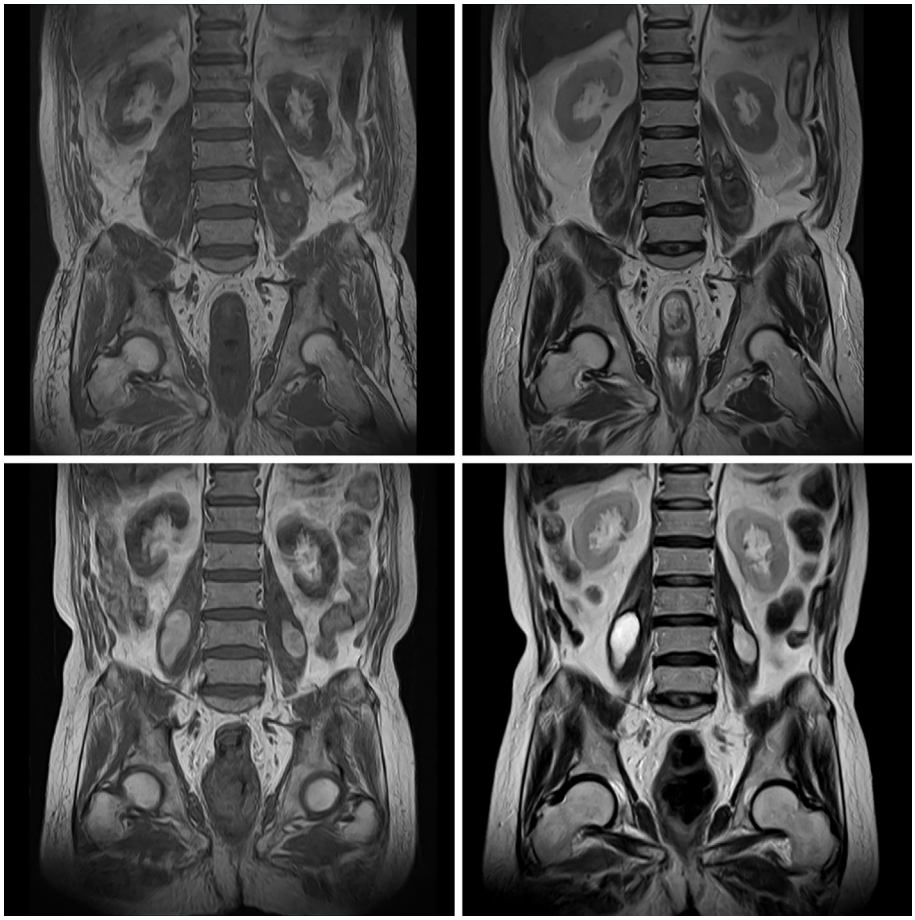

Figure 2. **Magnetic resonance imaging showed evidence of bilateral iliopsoas hematoma. T1-weighted images on the left side, T2-weighted images on the right side. MRI findings at Day 6 (upper row) revealed space occupying lesions (SOLs) in the bilateral iliopsoas with a low-intensity area surrounded by a high-intensity area on both T1- and T2-weighted imaging. MRI at Day 27 (lower row) showed high-intensity SOLs in the bilateral iliopsoas on both T1- and T1-weighted imaging, which was consistent with chronic hematoma.**

to correctly identify the cause of rhabdomyolysis and detect the presence of complications, including AKI, as the management of rhabdomyolysis and the patient's prognosis depend on these factors. For example, the prognosis of rhabdomyolysis complicated with AKI is considered to be worse than that of rhabdomyolysis without complication, as patients with rhabdomyolysis with AKI might require renal replacement therapy (4). Therefore, we should diagnose rhabdomyolysis before complications develop. Furthermore, we should promptly manage the cause of rhabdomyolysis and exacerbation factors, even if AKI is already complicated, in order to improve the prognosis. We should also perform appropriate and early aggressive repletion of fluids as the first step in managing rhabdomyolysis with AKI (1).

Bilateral iliopsoas hematoma is a rare cause of rhabdomyolysis complicated with AKI. The huge volume of hematoma in bilateral iliopsoas hematoma in the present case may have been due to the presence of rhabdomyolysis. The management of iliopsoas hematoma depends on the patient's hemodynamic status, comorbidities, and presence of active bleeding (12). If a patient with retroperitoneal hematoma, including iliopsoas hematoma, with rhabdomyolysis complicated by AKI requires renal replacement therapy, management of the retroperitoneal hematoma can become very complicated due to the need for an anticoagulant during renal replacement therapy (13). Fortunately, the present patient was able to avoid renal replacement therapy thanks to the speedy handling and careful treatment by the medical staff. Furthermore, if patients with iliopsoas hematoma have anemia, they might require blood transfusion. Rapid neutralization of anticoagulants, including warfarin and heparin, might be key to preventing patients with an iliopsoas hematoma from exacerbation of their condition and requiring blood transfusion.

In summary, we reported a rare case of bilateral hematoma complicated with rhabdomyolysis with AKI in a patient receiving warfarin therapy. We should pay close attention to episodes of turnover among elderly patients receiving warfarin therapy, as these patients are expected to become more and more frequent.

Written informed consent was obtained from the patient for

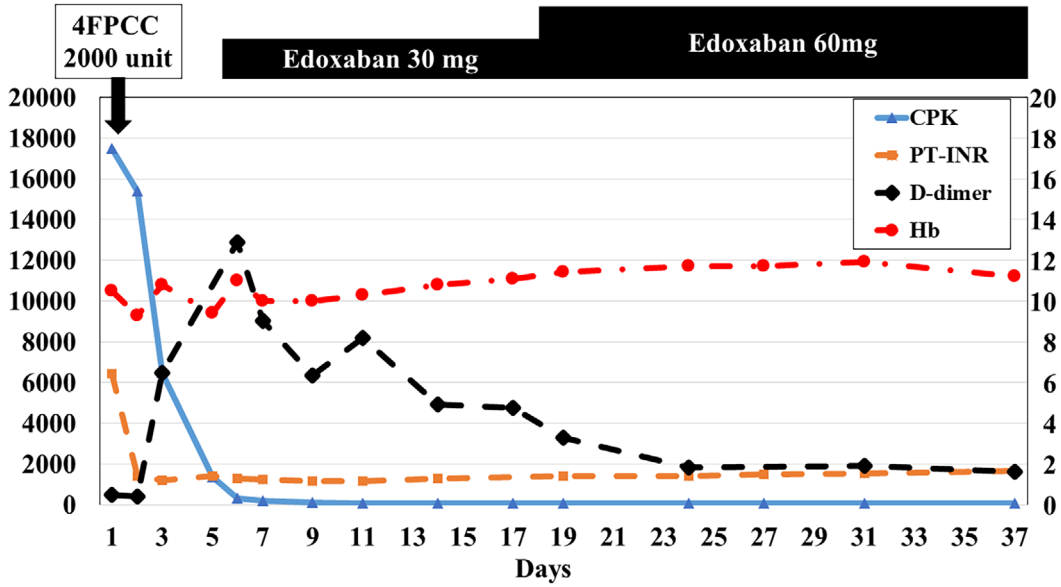

Figure 3. **Clinical course after hospitalization. The patient presented to the emergency department of our hospital with rhabdomyolysis and acute kidney injury suspected of being caused by bilateral iliopsoas hematoma. He was transferred to a tertiary medical hospital because his condition suggested that he should receive intensive care. He was administered four-factor prothrombin complex concentrates (4FPCC) to neutralize warfarin, and appropriate fluid infusion was begun; as a result, his clinical condition improved. Rhabdomyolysis and AKI also gradually ameliorated. We ultimately diagnosed him with rhabdomyolysis with AKI caused by bilateral iliopsoas hematoma. He underwent rehabilitation and was discharged without complications on Day 37.**

the publication of this case report and any accompanying images.

The authors state that they have no Conflict of Interest (COI).

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